

Exhibit U



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ASBESTOS MEDICAL EVALUATION

May 8, 1998

Boggs, Thomas J.
[REDACTED]

GA4/[REDACTED]

Gadsden, AL 35901

HISTORY: This is a 54 year old steelworker who reports intermittent exposure to asbestos materials since 1965 during his work in a steel plant. He worked in an office from 1965 to 1967. From 1968 until 1986 he worked as a supervisor in a plate mill. During this time he was involved with re-building furnaces about three times a year. He did hands-on work as well as supervisory work. As a result he had direct exposure to furnace insulation, pipe covering, cloth, gloves, gaskets, valve packing and firebrick. Since 1986 he has worked as a process systems engineer and product manager. He smoked one to one and one-half package of cigarettes daily for 33 years, quitting about a year and a half ago. Family history is non-contributory. He had a cholecystectomy in 1972 and knee surgery in 1978. Otherwise he has been healthy. Current medications are limited to Wellbutrin. On systems review he reports a lifelong dyspnea upon heavy exertion such as hard walking or manual labor. He has an occasional productive cough and rare chest pain that he attributes to gas.

PHYSICAL EXAM: Head and neck: No adenopathy or jugular venous distention. Chest: Symmetric expansion. No obvious chest wall deformities. Lungs: Normal palpation and percussion. Clear to auscultation anteriorly and posteriorly to the bases. Heart: Regular rhythm, without murmurs, clicks, rubs, or gallops. Extremities: No clubbing, cyanosis, or edema.

CHEST X-RAY: PA, lateral and bilateral oblique views of the chest dated 05/08/98 are reviewed for the presence of and classification of pneumoconiosis according to the ILO (1980). classification. Film quality is grade 1. Inspection of the lung parenchyma reveals a mild diffuse interstitial pattern, consisting of small irregular linear opacities within the lower lung zones bilaterally, of size and shape S/S, profusion 1/0. Examination of the pleural surfaces demonstrates slight circumscribed pleural thickening along the lateral thoracic walls, width A, extent 2 bilaterally. No pleural calcifications are observed. There are a few calcified nodules within the hilar structures which are most likely benign granulomas. No infiltrates, effusions or lung masses are present. The trachea is midline. The heart size is normal and the hilar structures are unremarkable. There are no other significant intrathoracic findings. No earlier films are available for comparison.

PULMONARY FUNCTION TESTING: Performed 05/08/98 using Crapo/Hsu predicted values. Forced vital capacity (FVC) is 4.73 liters (l.), or 89% predicted (pred.). FEV1 is 3.07 l. (74% pred.). FEV1/FVC ratio is 65%. FEF 25%-75% is 1.21 l./sec. (31% pred.). TLC is 8.45 l. (112% pred.). RV/TLC ratio is 43%. DICO is 60% pred., based on an IVC of 4.32 l. Inspection of the volume-time curves, flow-volume loops and diffusion graphs reveals good performance and reproducibility during those portions of the test. These pulmonary function tests demonstrate a mild obstructive defect with slight hyperinflation, air trapping and mildly reduced diffusion.

Boggs, Thomas J.
Page Two.

DIAGNOSIS/IMPRESSION: ^{1 2 3 4 5}

1. Pleural and parenchymal changes consistent with mild pulmonary asbestosis, given the environmental exposure history and latent period.
2. Mild chronic obstructive pulmonary disease (COPD). Clinical follow-up and a regular exercise program were recommended.

PROGNOSIS/RECOMMENDATION: Due to the latency period between exposure to asbestos and the onset of clinically significant asbestos-related disease, the patient is at increased risk for the development of bronchogenic carcinoma, mesothelioma, and other cancers, as well as for further deterioration in pulmonary function, even in the absence of additional asbestos exposure. Since these conditions may occur many years after exposure has terminated, close clinical follow-up, annual pulmonary re-evaluation, and continued avoidance of tobacco consumption are advisable.

This report relates only to the diagnosis of asbestos-related diseases, and is not intended to serve as a comprehensive evaluation of all health problems.

Jay T. Segarra, M.D.

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3. "Asbestos-related Disorders" In *Occupational Lung Disorders*, W. Raymond Parkes, ed.; Third Edition; Butterworth-Heinemann Ltd, London 1994.
4. Rom, William J.; "Asbestos-Related Diseases" In *Environmental & Occupational Medicine*, pp 72-77, 2d Ed, Little Brown & Co., 1982
5. Ernst P., Boueheen J. and Boddyk M.R. "Pleural Abnormality as a Cause of Impairment and Disability" In *The Third Wave of Asbestos Disease: Annals of the New York Academy of Sciences*, Volume 643, New York 1991.

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03-938-5

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June 7, 2004

Asbestos Clerk
Law Offices of Alwyn H. Luckey
P. O. Box 724
2016 Bienville Blvd.
Ocean Springs, MS 39566-0724

Re: Ball, Angelean

SSN: [REDACTED]

Chest radiograph(s) dated 10/15/99 is reviewed for the presence of and classification of pneumoconiosis (silicosis) according to the ILO 80 classification.

Film quality is grade 2 due to slight underexposure. Inspection of lung parenchyma demonstrates interstitial changes in all six lung zones, consisting of small rounded opacities of size and shape p/q, profusion 1/o.

There are no pleural plaques, pleural thickenings or pleural calcifications. No parenchymal infiltrates, nodules or masses are seen. The heart is of normal size and the mediastinal structures are unremarkable.

CONCLUSION: I have reviewed the occupational history and chest x-ray of the referenced individual. Based upon that history and the chest x-ray findings compatible with bilateral interstitial lung disease, it is my opinion, to a reasonable degree of medical certainty, that the x-ray changes are due to silicosis, acquired through occupational exposure to silica.

James W. Ballard, M.D.

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Jose E. Roman, M.D.

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July 26, 2002

ASBESTOS MEDICAL EVALUATION

JM-96

Eddie Lee King

Social Security Number: [REDACTED]

HISTORY:

I interviewed and examined Mr. King on 7/26/2002 for a medical evaluation for occupational lung disease.

This is a 57 year-old black male who reports a history of exposure to asbestos materials for 27 years. He refers shortness of breath on exertion, dry cough occasionally producing some sputum, and chest pains that have been already evaluated by a physician.

PAST MEDICAL AND OCCUPATIONAL HISTORY:

He refers a history of coronary artery disease with stents implanted in 1999 and in 2000, arterial hypertension, high cholesterol, and gastro-esophageal reflux disease.

He has history of smoking one half pack of cigarettes per day from 1969 until he quit in 1983.

He worked from 1969 to 1985 at Claiborne County Schools as welding teacher, from 1986 to 1988 at Owen Corning as fiberglass packer, from 1988 to 1996 with the City of Dallas as painter and sandblaster, and since 1996 he has been a substitute teacher with the Dallas Independent School System. He has been a self-employed remodeling contractor since 1989. He was exposed to asbestos working with insulation, pipe covering, asbestos gloves, asbestos cloth, tranxite, valve packing, asbestos gaskets, and firebrick in a dusty environment.

PHYSICAL EXAMINATION:

The subject is a well-developed black male, who is alert, oriented, and in no apparent distress. The chest shows symmetrical expansion and normal diaphragmatic excursion. The lungs are clear to auscultation and percussion. The heart has a regular rhythm, no murmur, and no gallops. The extremities show no clubbing, no cyanosis, and no edema.

PULMONARY FUNCTION TESTING:

Mr. King performed a PFT on 7/26/2002 using Crapo/Hsu predicted values for spirometry, lung volumes, and diffusion. The Forced Vital Capacity (FVC) is 3.84 Liters (L), (73% of predicted). The Forced Expiratory Volume in One Second (FEV1) is 2.84 L (70% of predicted); FEV1/FVC ratio is 74%. Forced Expiratory Flow 25%-75% is 2.07 Liters per second (55% of predicted). The Total Lung Capacity (TLC) is 5.91 L (78% of predicted), and the Residual Volume/TLC ratio is 35%. The diffusion single breath is 23.7 ml/min/mmHg (62% of predicted). This is compatible with mild restriction, small airways obstruction, and moderate decrease in diffusion.

CHEST RADIOGRAPHY:

Dr. James W. Ballard, NIOSH Certified B-Reader, evaluated the chest X-ray done on 7/25/2002. He classifies the patient as having evidence of parenchymal disease with

Asbestos Medical Evaluation
Eddie Lee King

Page 2

diffuse interstitial pattern consisting of small irregular linear opacities within the mid and lower lung zones bilaterally (ILO Size and Shape S/T, and Profusion 1/0). He has no evidence of asbestos-related pleural disease. There is cardiomegaly with aortic dilatation.

DIAGNOSTIC IMPRESSION:

1. Pulmonary Asbestosis. The patient has an exposure history to asbestos, restrictive physiology with a moderate diffusion defect in the PFT, and radiographic evidence of diffuse lung disease. The clinical and radiographic findings are compatible with the diagnosis of pulmonary asbestosis.
2. With the work history and my physical examination, I concur with Dr. James W. Ballard's B-reading radiographic evaluation.
3. Early obstructive lung disease; recommend clinical correlation by primary care physician.

PROGNOSIS AND RECOMMENDATIONS:

The history, clinical, and radiographic changes are compatible with early or mild pulmonary asbestosis. There is a high probability of deterioration in the pulmonary function and clinical picture, even in the absence of further asbestos exposure. This patient is at an increased risk for the development of bronchogenic carcinoma, mesothelioma, and other cancers, prominently in the gastrointestinal tract. The patient has a very high risk for the development of lung cancer, since the addition of both risks, tobacco smoking and asbestos exposure, will significantly increase the risk for developing lung cancer. Since these conditions may occur several years after the exposure has been terminated, close clinical follow up and annual pulmonary re-evaluation are recommended.

This report relates only to the diagnosis of asbestos-related lung disease, and is not intended to serve as a comprehensive evaluation of all health problems.



Jose E. Roman, M.D.

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